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Serial Left Ventricular Performance Evaluated by Cardiac Catheterization Before, Immediately After and at 6 Months After Balloon Aortic Valvuloplasty

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Although impaired ventricular function has been shown to improve after aortic valve replacement, there are few data on hemodynamic changes after balloon aortic valvuloplasty based on follow-up catheterization. Of 71 patients surviving 6 months after balloon aortic valvuloplasty, 41 agreed to late recatheterization. All patients had pre- and postvalvuloplasty and 6 month catheterization data measured with high fidelity micromanometer pressure recordings and simultaneous digital subtraction left ventriculography.

The hemodynamic result immediately after valvuloplasty included a reduction in the aortic valve gradient and a moderate increase in aortic valve area (0.51 ± 0.14 to 0.81 ± 0.19 cm^2, $p < 0.0001$). Ejection fraction increased slightly (52 ± 18 to 55 ± 17%, $p < 0.0001$) despite a decrease in peak positive rate of rise of left ventricular pressure (dP/dt 1,650 ± 460 to 1,500 ± 490 mm Hg/s, $p < 0.05$). There was also a decrease in left ventricular afterload and a small decrease in preload.

At 6 month recatheterization, the mean aortic valve gradient and area were similar to baseline values, with 31 (76%) of 41 patients demonstrating valvular restenosis. At 6 months many left ventricular hemodynamic variables, including peak positive dP/dt and stroke work, also resembled prevalvuloplasty values. However, left ventricular end-diastolic volume was reduced (111 ± 40 ml at 6 months versus 136 ± 52 ml before valvuloplasty, $p < 0.01$). The mean left ventricular ejection fraction was unchanged from prevalvuloplasty values in the study group of 41 patients, but was significantly improved in 9 of 15 patients with a baseline ejection fraction <50%.

Thus, recatheterization at 6 months demonstrates left ventricular remodeling and a modest improvement in ventricular performance, with the left ventricle being less dilated while maintaining stroke work and cardiac output. These changes occur despite a high incidence of valve restenosis, indicating that balloon aortic valvuloplasty should be reserved for patients unable to tolerate surgical valve replacement.

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Clinical symptoms in patients with aortic stenosis usually result from left ventricular dysfunction secondary to the valvular stenosis. Left ventricular dysfunction has been shown to improve and in some cases reverse after surgical aortic valve replacement (1–3). Unfortunately, many patients with aortic stenosis are elderly and have coexisting medical illness, thus increasing the operative risk for aortic valve replacement (4). An operative mortality rate of 10.8% was reported by Rioux et al. (5) in patients >70 years of age undergoing aortic valve replacement, whereas Edmunds et al. (6) reported an operative mortality rate of about 30% in octogenarians. With medical treatment, however, the prognosis with this disease is dismal. O'Keefe et al. (7) reported a 1 and 2 year survival rate of 57% and 37%, respectively, in patients with severe aortic stenosis treated medically because of excessive surgical risk or refusal of surgery.

During the past several years, balloon aortic valvuloplasty has been employed as an alternative treatment for high surgical risk patients with critical aortic stenosis (8). Although many patients demonstrate initial critical improvement, recurrence of symptoms as early as 6 months after the

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procedure has been common. Valvular restenosis has not been infrequent and may be demonstrated noninvasively by Doppler echocardiography (9). Echocardiography, however, is limited in its ability to define changes in left ventricular function after balloon aortic valvuloplasty. Left ventricular performance in patients with aortic stenosis is often responsible for clinical symptoms and, in our experience (10), reduced ejection fraction before balloon aortic valvuloplasty is the best predictor of clinical outcome after valvuloplasty.

The purpose of this study was to define the complex hemodynamic changes that occur immediately after balloon aortic valvuloplasty and to reevaluate left ventricular performance in these patients 6 months after the procedure.

Methods

Study patients. The study group consisted of 112 consecutive patients undergoing percutaneous balloon aortic valvuloplasty between November 1986 and January 1989 at Duke University Medical Center. All patients had symptomatic aortic stenosis, with an initial aortic valve area <0.8 cm^2. Each patient was evaluated by a cardiologist and cardiovascular surgeon. Ten patients had refused aortic valve surgery and 102 were considered to have a high risk for morbidity or mortality from aortic valve replacement. High surgical risk was due to age >75 years in 68 patients, left ventricular ejection fraction <30% in 22, severe obstructive pulmonary disease in 4, dementia in 4, malignancy in 3 and chronic renal failure in 1. All patients signed informed consent for the study, which had been previously approved by the investigational review board of our institution.

Clinical status was assessed by New York Heart Association standards for congestive heart failure and the Canadian classification for angina. All patients had 6 month clinical follow-up data. Of the 112 patients, 31 died and 10 underwent subsequent aortic valve replacment before the 6 month follow-up study. The 10 patients who underwent subsequent aortic valve replacement included 3 who had refused surgery and subsequently agreed, 3 who originally required urgent noncardiac surgery and then underwent valve replacement, 2 with a low ejection fraction that improved after balloon aortic valvuloplasty and 2 who, despite continued high surgical risk, underwent surgical valve replacement because of continued severe symptoms of congestive heart failure. All 10 patients survived operation without major complication.

Patients eligible for 6 month recatheterization (Tables 1 and 2). Of the 71 patients who survived 6 months after balloon aortic valvuloplasty, 41 (58%) agreed to undergo late recatheterization at mean of 6 ± 1.2 months after the procedure (Fig. 1). Of these 41 patients, 23 (56%) were clinically improved and 18 (44%) had recurrence of prevalvuloplasty symptoms at the time of recatheterization. The baseline clinical data in the 112 original patients with aortic valvuloplasty and the 41 patients with 6 month recatheterization are compared in Table 1. The 41 patients undergoing recatheterization were similar with respect to age, gender, extent of coronary artery disease and symptoms of congestive heart failure and angina. The baseline hemodynamic variables of the original 112 patients and the 41 undergoing recatheterization were similar as well (Table 2).

Table 1. Comparison of Baseline Clinical Variables of the 112 Patients Undergoing Balloon Aortic Valvuloplasty (BAV) and 41 Patients Undergoing Late Recatheterization (Recath)

	Patients With 6 Month Recath (n = 41)	All BAV Patients (n = 112)
Age (yr)	75.9 ± 8.2	75.9 ± 7.7
% male	42	37
Extent of CAD*		
None	20 (48%)	49 (44%)
1 vessel	11 (27%)	30 (27%)
2 vessel	6 (15%)	19 (17%)
3 vessel	4 (10%)	14 (12%)
CHF (NYHA class)		
I	5 (12%)	8 (7%)
II	3 (7%)	12 (11%)
III	17 (42%)	40 (36%)
IV	16 (39%)	52 (46%)
Angina (Canadian class)		
0-I	17 (41%)	47 (42%)
II	6 (15%)	11 (10%)
III	11 (27%)	30 (27%)
IV	7 (17%)	24 (21%)

There were no statistical differences in any of the values between the two groups. CAD = coronary artery disease; CHF = congestive heart failure; NYHA = New York Heart Association.

Hemodynamic evaluation. Hemodynamic measurements were obtained immediately before and after valvuloplasty,

Table 2. Comparison of Baseline Hemodynamic Variables in 112 Patients Undergoing Balloon Valvuloplasty (BAV) and 41 Patients Undergoing Late Recatheterization (Recath)

	Patients With 6 Month Recath* (n = 41)	All BAV Patients (n = 112)
Mean aortic valve gradient (mm Hg)	56.6 ± 20.1	59.7 ± 18.4
Baseline aortic valve area (cm^2)	0.52 ± 0.16	0.51 ± 0.14
Change in aortic valve area with BAV (cm^2)	0.27 ± 0.14	0.29 ± 0.14
Cardiac output (liters/min)	4.1 ± 1.3	4.3 ± 1.1
Baseline LVEF (%)	46.6 ± 19.4	51.9 ± 18.0
LVEF <50% before BAV	15 (37%)	50 (45%)
Baseline end-diastolic volume (ml)	141 ± 58	137 ± 51
Baseline end-systolic volume (ml)	79 ± 56	68 ± 49

There were no statistical differences in any of the values between the two groups. LVEF = left ventricular ejection fraction.

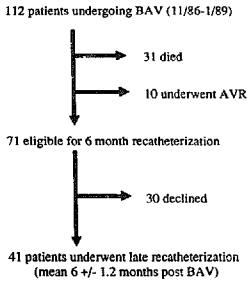

Figure 1. Flow diagram illustrating the clinical status of the patients with balloon aortic valvuloplasty (BAV) 6 months after the procedure. AVR = aortic valve replacement.

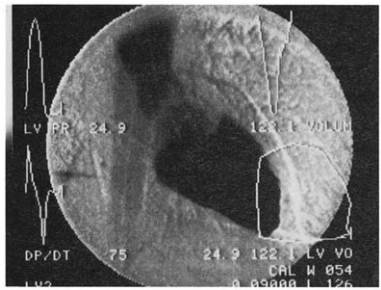

Figure 2. Digital subtraction left ventriculogram showing the digitized, simultaneous left ventricular pessure waveform (LVPR) at **left center**. The left ventricular volume curve is shown at **right center** and the rate of change of left ventricular pressure (dP/dt) is shown at **bottom left**. The simultaneous left ventricular pressure-volume data generating the pressure-volume loop are shown at **bottom right**.

as well as at the time of late recatheterization (6 ± 1.2 months after valvuloplasty). Cardiac catheterization was performed by means of the femoral route.

Right heart pressures and hemodynamic variables were obtained at all three catheterization studies: before and immediately after valvuloplasty and at the 6 month study. Coronary arteriography was performed at the initial assessment in all patients. Aortic regurgitation was evaluated by supravalvular aortography at each of the three examinations and was graded from 0 to 4 using a standard visual grading scale (11). To define the hemodynamic variables as accurately as possible, simultaneous left ventricular and aortic pressure measurements were made with use of a high fidelity, dual sensor micromanometer pigtail catheter (Millar Instruments) positioned across the aortic valve. Oxygen consumption was directly measured with use of a metabolic cart (Waters instrument or Sensor Medics MMC Horizon), allowing determination of the Fick cardiac output from the arteriovenous oxygen difference. The Gorlin formula was used to calculate aortic valve area (12).

Digital subtraction left ventriculograms were obtained in the 30° right anterior oblique projection with use of a nonionic contrast agent (Isovue [iopamidol], E. R. Squibb & Sons or Omnipaque [iohexol], Winthrop, Pharmaceuticals). A General Electric MLX L/U radiographic X-ray system coupled to an ADAC 4100-C computer was used for image processing. Left ventricular and aortic pressures were digitized and stored at 5 ms intervals. Left ventriculograms were obtained by injecting 45 ml of contrast agent at 12 ml/s, with images recorded at 30 frames/s. The area-length method was used to calculate left ventricular volumes and ejection fraction (13). Volumes obtained in this laboratory have been previously standardized against left ventricular casts of known volume, generating a regression equation from which volumes were determined. Pressure-volume loops were constructed with use of the simultaneous pressure and volume data (Fig. 2). Areas defined by these loops were used to calculate stroke work.

Balloon valvuloplasty procedure. The aortic valvuloplasty balloon (Mansfield) was positioned retrogradely across the aortic valve through a 14F sheath in the femoral artery. A single balloon of 20 mm diameter was most commonly used. The balloon was inflated until the waist in the balloon was seen to disappear; the balloon was inflated two to five times at 3 to 8 atm of pressure.

Statistical analysis. The data are expressed as mean values ± SD unless otherwise indicated. Statistical analysis was performed with use of the Wilcoxon signed rank test for changes in hemodynamic variables. Statistical significance was defined at $p < 0.05$.

Results

Hemodynamic changes immediately after valvuloplasty (Tables 3 and 4). Immediately after aortic valvuloplasty, there was a decrease in mean aortic valve gradient from 59.7 ± 18.4 to 35.6 ± 12.7 mm Hg ($p < 0.01$). There was no change in Fick cardiac output. The calculated aortic valve area increased from 0.51 ± 0.14 to 0.81 ± 0.19 cm^2 ($p < 0.01$). Left ventricular systolic pressure was reduced from 215 ± 36 to 182 ± 31 mm Hg ($p < 0.01$). There was no significant change in aortic systolic or diastolic pressure. Left ventricular end-diastolic pressure decreased from

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Table 3. Hemodynamic Variables Related to Aortic Valve Area Before (Pre) and After (Post) Balloon Aortic Valvuloplasty (BAV) and at 6 Month Recatheterization (Recath) in 41 Patients

	Pre BAV	Post BAV	6 Month Recath
Mean aortic gradient (mm Hg)	59.7 ± 18.4	35.6 ± 12.7*	53.1 ± 16.5*
Cardiac output (liters/min)	4.3 ± 1.1	4.5 ± 1.2	4.4 ± 1.2
Aortic valve area (cm^2)	0.51 ± 0.14	0.81 ± 0.19*	0.58 ± 0.16*
LV systolic pressure (mm Hg)	215 ± 36	182 ± 31*	211 ± 40
Aortic systolic pressure (mm Hg)	146.2 ± 33.3	145 ± 33	155.7 ± 31.1
Aortic diastolic pressure (mm Hg)	64 ± 13	63 ± 16	64 ± 13

*$p < 0.01$ compared with before balloon aortic valvuloplasty. LV = left ventricular.

19.3 ± 7.8 to 15.3 ± 7.6 mm Hg ($p < 0.01$) whereas there was no change in end-diastolic volume. There was a small decrease in left ventricular end-systolic volume from 66.2 ± 48.6 to 62.7 ± 46.2 ml ($p = 0.02$) and a small increase in ejection fraction from 51.9 ± 18 before to 55 ± 17.3% after valvuloplasty ($p < 0.01$).

The magnitude of both the peak positive and the peak negative maximal rate of rise of left ventricular pressure (dP/dt) decreased immediately after valvuloplasty. There was a decrease in stroke work from 16.3 ± 6.9 × 10^6 to 14 ± 5.1 × 10^6 ergs ($p < 0.01$). There was no change in right atrial pressure, pulmonary artery pressure or pulmonary capillary wedge pressure either immediately after valvuloplasty or at the time of 6 month recatheterization.

Table 4. Indexes of Left Ventricular (LV) Performance Before (Pre) and After (Post) Balloon Aortic Valvuloplasty (BAV) and at 6 Month Recatheterization (Recath)

	Pre BAV	Post BAV	6 Month Recath
LVEF (%)	51.9 ± 18	55 ± 17.3*	54.4 ± 15.6
LV end-diastolic volume (ml)	136 ± 52	134 ± 51	111 ± 40*
LV end-systolic volume (ml)	66.2 ± 48.6	62.7 ± 46.2*	54 ± 36
LV end-diastolic pressure (mm Hg)	19.3 ± 7.8	15.3 ± 7.6*	19.5 ± 8.7
Stroke work (× 10^6 ergs)	16.3 ± 6.9	14 ± 5.1*	13.8 ± 5.9
Peak positive dP/dt (mm Hg/s)	1,650 ± 460	1,500 ± 490*	1,750 ± 490
Peak negative dP/dt (mm Hg/s)	−1,440 ± 370	−1,290 ± 410*	−1,450 ± 670
Heart rate (beats/min)	74.4 ± 15.5	76.9 ± 15.9	76.5 ± 17.1

*$p < 0.01$ compared with before balloon aortic valvuloplasty. dP/dt = maximal rate of rise of left ventricular pressure; LVEF = left ventricular ejection fraction.

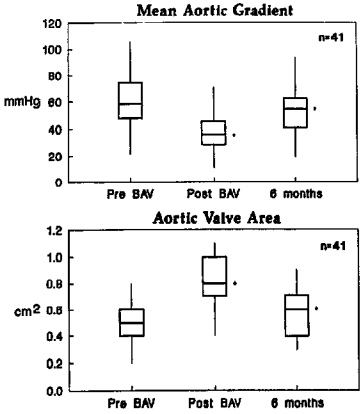

Figure 3. Top, Mean aortic gradient and aortic valve area before (Pre) and immediately after (Post) balloon aortic valvuloplasty (BAV) and at 6 month recatheterization in the 41 patients, illustrated by box and whisker plots (25). (The box shows the median as well as the 25th and 75th quartiles of the data; the whiskers show the range of the data). The aortic valve gradient (**top**) was significantly lower immediately after valvuloplasty. Much of the pressure gradient had returned at the 6 month recatheterization, although it remained less than that before valvuloplasty. **Bottom,** The aortic valve area increased immediately after balloon aortic valvuloplasty; its return toward its prevalvuloplasty value is evident at 6 months. *Differences significant at $p < 0.05$.

Hemodynamic results at the time of late recatheterization. At this examination, the mean aortic gradient had returned toward the baseline value, although it remained slightly less than that before valvuloplasty (53.1 ± 16.5 versus 59.7 ± 18.4 mm Hg, $p < 0.01$). The calculated aortic valve area often demonstrated significant restenosis, but the mean valve area remained slightly larger than that before valvuloplasty (0.58 ± 0.16 versus 9.51 ± 0.14 cm^2, $p < 0.01$) (Fig. 3). The Fick cardiac output and heart rate were unchanged at each interval.

Left ventricular peak systolic pressure at late recatheterization was not different than that before valvuloplasty, although there was a trend toward higher aortic systolic pressure at 6 month recatheterization (155.7 ± 31.1 versus 146.2 ± 33.3 mm Hg, $p = 0.057$). There was no difference in the aortic diastolic pressure.

Although left ventricular end-diastolic pressure was un-

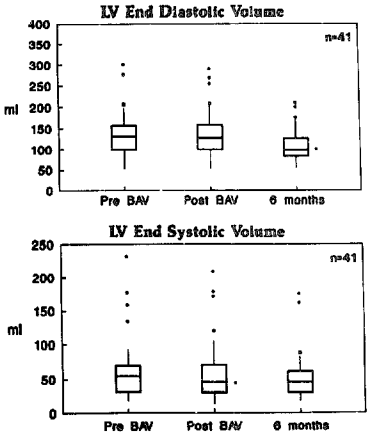

Figure 4. A, Box and whisker plots of the left ventricular end-diastolic volume (top) and end-systolic volume (bottom) at the three times of study. The outliers in the data are shown as individual points (25). A reduction in left ventricular end-diastolic volume is evident at the 6 month recatheterization. B, The end-systolic volume was slightly reduced immediately after balloon aortic valvuloplasty (Post BAV). At the 6 month recatheterization the end-systolic volume was not different from that before valvuloplasty (Pre BAV). *Differences significant at $p < 0.05$ compared with pre BAV.

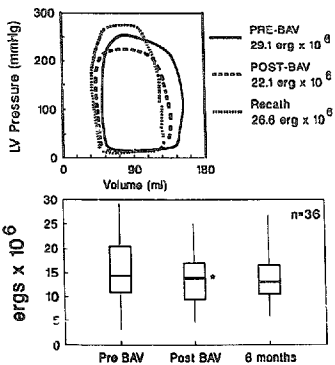

Figure 5. Top, Left ventricular (LV) pressure-volume loops at the three study times from a representative patient after balloon aortic valvuloplasty (Post BAV). Left ventricular stroke work is calculated from the area of the pressure-volume loop. Bottom, Box and whisker plots of the left ventricular stroke work data from the three study times in 36 patients. Stroke work was reduced immediately after valvuloplasty (Post BAV) (* = $p < 0.05$). At the 6 month recatheterization (Recath), there was a trend toward reduced stroke work ($p = 0.095$ compared with before valvuloplasty [Pre BAV]).

changed at 6 months compared with before valvuloplasty, there was a significant decrease in left ventricular end-diastolic volume at the time of the late recatheterization (from 136 ± 52 to 111 ± 40 ml, $p < 0.01$) (Fig. 4, top). Of the 41 patients undergoing late recatheterization, 29 (71%) demonstrated a decrease in left ventricular end-diastolic volume compared with that before valvuloplasty. There was a trend toward a reduction in end-diastolic volume in those patients with a more dilated left ventricle before valvuloplasty ($r = 0.61$). The change in left ventricular end-diastolic volume did not correlate with prevalvuloplasty measurements of left ventricular ejection fraction, end-diastolic pressure and aortic valve gradient and area. It also did not correlate with changes in ejection fraction, end-diastolic pressure and aortic valve gradient and valve area assessed at late recatheterization.

The end-systolic volume at late recatheterization was not different from the value before valvuloplasty (Fig. 4, bottom). The magnitude of both peak positive and peak negative dP/dt was not different from that before valvuloplasty. There was a trend toward decreased stroke work at the 6 month recatheterization (13.8 ± 5.9 × 10^6 versus 16.3 ± 6.9 × 10^6 ergs, $p = 0.095$) (Fig. 5).

At 6 months, the left ventricular ejection fraction was similar to the prevalvuloplasty value, although some patients with a depressed prevalvuloplasty ejection fraction demonstrated significant improvement at 6 months (Fig. 6). Indeed, of 15 patients with a baseline ejection fraction <50%, 9 had an increase after the 6 month interval.

Discussion

This study represents the largest group of patients for whom invasive hemodynamic data evaluating left ventricular performance are available both immediately and at late follow-up study after balloon aortic valvuloplasty. Immediately after valvuloplasty, moderate improvement in the severity of aortic stenosis was achieved, similar to the initial results of others (14–16).

Acute changes in ventricular performance. Immediately after valvuloplasty, the increase in the aortic orifice size causes a reduction in left ventricular afterload. This reduction in afterload results in an immediate decrease in end-systolic volume and contributes to the small increase in

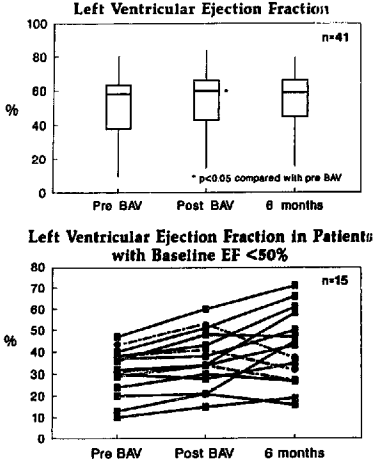

Figure 6. Top, Box and whisker plots of left ventricular ejection fraction (EF) before valvuloplasty (Pre BAV), immediately after valvuloplasty (Post BAV) and at 6 month recatheterization in the 41 patients. A slight increase in ejection fraction occurred immediately after valvuloplasty (Post BAV), but at 6 months it was similar to the prevalvuloplasty value. **Bottom,** Left ventricular ejection fraction in the 15 patients with a depressed prevalvuloplasty ejection fraction (<50%). Nine patients demonstrated significant improvement in ejection fraction at the 6 month recatheterization. **Dashed lines** connect patients with no improvement or worsening of ejection fraction.

ejection fraction observed immediately after balloon aortic valvuloplasty (17).

The increase in ejection fraction occurs despite a decrease in peak positive left ventricular dP/dt that is likely caused by ischemic stunning of the ventricle, resulting from the decrease in coronary perfusion pressure that occurs transiently during balloon inflation. This transient ischemia during balloon inflation, on occasion, results in overt angina and ischemic electrocardiographic changes. Further evidence for left ventricular ischemia comes from data showing a reduction in the rate of isovolumetric relaxation immediately after aortic valvuloplasty (18). In addition, coronary sinus measurements have demonstrated a decrease in coronary blood flow and a change to myocardial lactate production for a period of 5 to 10 min after balloon inflation (19).

The end-diastolic volume or preload of the left ventricle immediately after the procedure was similar to the prevalvuloplasty value, with only a slight decrease in end-diastolic pressure. There was a decrease in left ventricular stroke work immediately after valvuloplasty, primarily the result of the lower systolic pressure the left ventricle was required to generate. The immediate effect of aortic valvuloplasty are therefore complex, with an interplay of factors that affect left ventricular loading conditions.

In addition, it has been postulated that catecholamine release after balloon inflation might augment intrinsic myocardial contractility and that left ventricular baroreceptor stimulation might reduce systemic arterial resistance and further reduce afterload immediately after the procedure. The similarity of the heart rate before and after the procedure, however, and the evidence that the decrease in aortic valve resistance contributes the primary component to the reduction in left ventricular afterload observed immediately after the procedure suggest that these two latter factors contribute little to the hemodynamic outcome observed immediately after balloon aortic valvuloplasty (20).

Aortic valve restenosis. The 6 month recatheterization data demonstrate that restenosis of the aortic valve is frequent. Other investigators have reported varying results with respect to late restenosis after aortic valvuloplasty. Letac et al. (16) found only 9 (24%) of 37 patients with late valvular restenosis. Restenosis in their series was defined as a loss of ≥50% of the increase in valve area observed immediately after valvuloplasty. These data were in an extremely select group of 37 patients from a total of 218 patients. Subsequent reported studies have not found such an optimistic rate of late aortic valve restenosis. Grollier et al. (21), studying 24 patients by recatheterization only 8 days after valvuloplasty, found 16 (66%) of 24 patients to have valvular restenosis. In our study, in which restenosis was defined as a 50% loss in the initial improvement in valve area achieved by aortic valvuloplasty, 31 (76%) of 41 patients had valvular restenosis at 6 months.

The pathologic anatomy of these valves may help explain the high rate of restenosis (22). The cause of stenosis in this elderly group of patients is usually degenerative calcific disease of a tricuspid aortic valve. The valve cusps have gross calcific deposits that lie in the valve sinuses and impede opening of the valve. Commissure fusion contributes only a small amount to orifice narrowing, unlike the pathology in rheumatic aortic stenosis. The increase in valve area produced by balloon aortic valvuloplasty in degenerative calcific aortic stenosis has been shown to be due to gross fracture of the nodular calcium plaques, occasional separation of fused commissures and possibly some stretching of the aortic anulus (23). Thus, the underlying valve pathology and the limited alteration possible with balloon valvuloplasty help explain the moderate improvement in valve area seen immediately and the high rate of late valvular restenosis.

Left ventricular performance at 6 months. Many of the variables of left ventricular performance assessed at the time of late recatheterization were similar to the prevalvuloplasty values. Left ventricular systolic and end-diastolic pressures were unchanged from prevalvuloplasty values. The mean left ventricular ejection fraction at late recatheterization was also not significantly different from that before valvuloplasty. However, in some patients ejection fraction was depressed before valvuloplasty but showed significant improvement at late recatheterization (Fig. 6, bottom). Similarly, Safian et al. (24) found that a subset of their patients with a depressed left ventricular ejection fraction (37 ± 11%) demonstrated significant improvement in this variable when reevaluated by radionuclide ventriculography 3 months after aortic valvuloplasty.

Reduction in ventricular dilation at 6 months. Additionally, our late recatheterization data demonstrate a significant reduction in the mean left ventricular end-diastolic volume at 6 months. There was a trend for the greatest reduction to occur in patients with the greatest left ventricular dilation before valvuloplasty. These data are supported by the reduction in end-diastolic volume demonstrated by radionuclide ventriculography 3 months after balloon aortic valvuloplasty (24). Thus, 6 months after balloon aortic valvuloplasty, remodeling of the left ventricle appears to be evident. Despite restenosis, the left ventricle is less dilated and is able to maintain cardiac output at a reduced preload. With the ventricle being less dilated, diastolic wall stress is likely reduced, decreasing myocardial oxygen demand.

Whether this subtle improvement in ventricular performance may partially explain the clinical improvement seen in some patients after balloon aortic valvuloplasty needs further evaluation. Improvement in diastolic ventricular function (both in terms of the time course of isovolumetric relaxation [tau] and left ventricular compliance during passive filling [Kp]), were recently shown (18) to correlate with clinical improvement after balloon aortic valvuloplasty.

Clinical implications. The high rate of restenosis suggests that improvement in ventricular performance is not likely to persist. In addition, the 6 month mortality rate after balloon aortic valvuloplasty is high (28% in our study). These data present further evidence that aortic valve replacement should remain the treatment of choice for operative candidates with aortic stenosis. Balloon aortic valvuloplasty remains a palliative procedure that should be strictly reserved for those patients who are unable to tolerate aortic valve replacement. The procedure may also benefit the subset of patients with a high operative risk due to depressed left ventricular ejection fraction. Aortic valvuloplasty may be used as a "bridge" procedure to potentially improve the ejection fraction and clinical status in some of these patients and possibly decrease their subsequent operative risk. The ability to predict which patients will demonstrate an improvement in ejection fraction after the procedure would be extremely useful and needs further investigation.

Limitations of the study. The 41 patients undergoing late recatheterization in this study represent a biased group of patients because it does not include those patients who did not survive for 6 months. The clinical hemodynamic data of the entire group of 112 patients and the 41 undergoing late restudy were similar, however, and we believe that the hemodynamic data of the 41 patients undergoing late recatheterization are representative of the entire group of patients having valvuloplasty.

Late hemodynamic data in all patients surviving 6 months after valvuloplasty would be optimal. Because of this elderly and often extremely ill group of patients, a 58% recatheterization rate is higher than in any previous study.

Conclusions. The hemodynamic changes occurring immediately after valvuloplasty are complex and include a decrease in afterload, little change in preload, a moderate increase in left ventricular ejection fraction and probable ischemic "stunning" of the ventricle.

Recatheterization of patients 6 months after balloon aortic valvuloplasty reveals significant restenosis in most patients as judged by recurrence of a pressure gradient and near return of valve area to its prevalvuloplasty level. Many variables of left ventricular performance were similar to prevalvuloplasty values. Although overall the left ventricular ejection fraction was not different from values before valvuloplasty, in some patients a depressed prevalvuloplasty ejection fraction demonstrated significant improvement at 6 months. In addition, at 6 month recatheterization, remodeling of the left ventricle is evident, with the left ventricle able to maintain stroke work and cardiac output at a reduced end-diastolic volume despite evidence of valvular restenosis.

Modest improvement in ventricular performance 6 months after balloon aortic valvuloplasty may be responsible for clinical improvement. However, the restenosis evident at the 6 month recatheterization indicates that the improvement in left ventricular performance is unlikely to persist and that balloon aortic valvuloplasty should continue to be performed only as a palliative procedure for those patients with symptomatic aortic stenosis who are unable to tolerate aortic valve replacement.

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References

1. Smith N, McAnulty JH, Rahimtoola SH. Severe aortic stenosis with impaired left ventricular function and clinical heart failure: results of valve replacement. Circulation 1978;58:255–64.
2. Croke RP, Pifarre R, Sullivan H, Gunnar R, Loeb H. Reversal of advanced left ventricular dysfunction following aortic valve replacement for aortic stenosis. Ann Thorac Surg 1977;24:38–43.

3. Murphy ES, Lawson RM, Starr A, Rahimtoola SH. Severe aortic stenosis in patients 60 years of age and older: left ventricular function and 10 year survival after valve replacement. Circulation 1981;64(suppl II):II-184–8.

4. Maguvern JA, Pennock JL, Campbell DB, et al. Aortic valve replacement and combined aortic valve replacement and coronary artery bypass grafting: predicting high risk groups. J Am Coll Cardiol 1987;9:38–43.

5. Rioux C, Logeais Y, Leguerrier A, et al. Valvular replacement for aortic stenosis in patients over 70 years: immediate risk and long-term results (from a consecutive series of 355 patients). Eur Heart J 1988;9(suppl E):121–7.

6. Edmunds LH, Stephenson LW, Edie RN, Ratcliffe MB. Open-heart surgery in octogenarians. N Engl J Med 1988;319:131–6.

7. O'Keefe JH, Vlietstra RE, Bailey KR, Holmes DR. Natural history of candidates for balloon aortic valvuloplasty. Mayo Clin Proc 1987;62:986–91.

8. Cribier A, Saoudi N, Berland J, Savin T, Rocha P, Letac B. Percutaneous transluminal valvuloplasty of acquired aortic stenosis in elderly patients: an alternative to valve replacement? Lancet 1985;1:63–7.

9. Come PC, Riley MF, Safian RD, Ferguson JF, Diver DD, McKay RG. Usefulness of noninvasive assessment of aortic stenosis before and after percutaneous aortic valvuloplasty. Am J Cardiol 1988;61:1300–6.

10. Davidson CJ, Harrison JK, Leithe ME, Kisslo KB, Bashore TM. Failure of balloon aortic valvuloplasty to result in sustained clinical improvement in patients with depressed left ventricular function. Am J Cardiol 1990; 65:72–7.

11. Paulin S. Aortography. In: Grossman W, ed. Cardiac Catheterization and Angiography. Philadelphia: Lea & Febiger, 1986:227–47.

12. Gorlin R, Gorlin G. Hydraulic formula for calculation of area of stenotic mitral valve, other cardiac valves, and central circulatory shunts. Am Heart J 1951;41:1–10.

13. Dodge HT, Sandler H, Ballew DW, Lord JD Jr. The use of biplane angiocardiography for the measurement of left ventricular volume in man. Am Heart J 1960;60:762–76.

14. McKay RG, Safian RD, Lock JE, et al. Assessment of left ventricular and aortic valve function after aortic balloon valvuloplasty in adult patients with critical aortic stenosis. Circulation 1987;75:192–201.

15. Safian RD, Berman AD, Diver DJ, et al. Balloon aortic valvuloplasty in 170 consecutive patients. N Engl J Med 1988;319:125–30.

16. Letac B, Cribier A, Koning R, Bellefleur JP. Results of percutaneous valvuloplasty in 218 adults with valvular aortic stenosis. Am J Cardiol 1988;62:598–605.

17. Harrison JK, Honemann JD, Leithe ME, Davidson CJ, Kisslo K, Bashore TM. Acute and chronic measures of left ventricular contractile performance, corrected for afterload, are unchanged following balloon aortic valvuloplasty (abstr). J Am Coll Cardiol 1990;15:42A.

18. Sheikh KH, Davidson CJ, Honan M, Kisslo K, Skelton TN, Bashore TM. Clinical status 6 months after aortic valvuloplasty relate to changes in left ventricular diastolic performance and mass (abstr). Circulation 1989, 80(suppl II):II-360.

19. Rousseau MF, Wyns W, Hammer F, Caucheteux D, Hue L, Pouleur H. Changes in coronary blood flow and myocardial metabolism during aortic balloon valvuloplasty. Am J Cardiol 1988;61:1080–4.

20. Kass D, Davidson C, Skelton TM, Bashore TM. Altered LV afterload due to aortic valve: an alternative to valve area for assessing valvuloplasty outcome (abstr). Circulation 1988;78(suppl II):II-52B.

21. Grollier G, Commeau P, Sesboue B, Huret B, Potier JC, Foucault JP. Short-term clinical and haemodynamic assessment of balloon aortic valvuloplasty in 30 elderly patients: discrepancy between immediate and eight-day haemodynamic values. Eur Heart J 1988;155–62.

22. Waller BF, McKay CR, Erny R, Morgan R, Mohler E. Catheter balloon valvuloplasty of necropsy stenotic aortic valves: etiology of aortic stenosis as a major factor in early restenosis (abstr). J Am Coll Cardiol 1989;13:16A.

23. McKay RG, Safian RD, Lock JE, et al. Balloon dilatation of calcific aortic stenosis in elderly patients: postmortem, intraoperative, and percutaneous valvuloplasty studies. Circulation 1986;74:119–25.

24. Safian RD, Warren SE, Berman AD, et al. Improvement in symptoms and left ventricular performance after balloon aortic valvuloplasty in patients with aortic stenosis and depressed left ventricular ejection fraction. Circulation 1988;78:1181–91.

25. Williamson DF, Parker RA, Kendrick JS. The box plot: a simple visual method to interpret data. Ann Intern Med 1989;110:916–21.